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RACE AND OXIDATIVE STRESS MARKERS DETERMINE BLOOD PRESSURE VARIABILITY IN INDIVIDUALS WITH HIGH CARDIOVASCULAR DISEASE RISK

ACC Moderated Poster Contributions
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Background: Oxidative stress (OS) and cardiovascular reactivity are related to cardiovascular (CV) morbidity and mortality. However, little is known about the relationships between these cardiovascular risk factors and their confounders. We hypothesize that higher OS is linked to higher blood pressure (BP) reactivity to laboratory stressors and in natural setting

Methods: 137 individuals with family history of hypertension and early myocardial infarction were studied. There were 63 Whites (38 males) and 74 Blacks(35 males),aged 19 to 36(27.6+/-3.1). Each underwent a protocol including a competitive video game (VG), cold stressor, and ambulatory BP recording. BP and HR were measured and blood samples were drawn 6 times throughout the protocol to assay 8-OHdG and 8-Isoprostane as DNA damage and lipids peroxidation markers. Repeated measures analyses of covariance were used to test for mean differences (controlling for BMI) and Pearson correlations were used to test associations between OS and BP.

Results: There were no significant race differences in BP reactivity to either stressor (both p's>0.48). 8-OHdG levels were significantly lower across all time points for Blacks than for Whites ($p<0.05$) while levels of 8-isoprostane did not differ significantly ($p>.10$). 8-OHdG levels averaged across all time points were significantly correlated with SBP reactivity ($r=0.45$, <0.01) and 24-hour, daytime, and nighttime SBP (r range=0.37 to 0.42, all p's<0.02) for Whites but not for Blacks, whereas 8-isoprostane levels were significantly correlated with reactive SBP and nighttime DBP (both $r=0.38$, $p<0.01$)for Blacks but not for Whites.

Conclusions: These findings suggest a link between OS and BP changes in individuals at high CV disease risk. Further, race determine which OS marker will impact blood pressure variation implying race differences in OS related mechanisms of CVD